

# Acquired aortic regurgitation following occlusion of the persistent arterial duct: an echocardiographic assessment

J R Harrisberg, K Govendrageloo, V Hunter, S E Levin

## Abstract

**Objective**—To document the development of aortic regurgitation following occlusion of a patent arterial duct.

**Design**—Case series involving nine children referred for surgical ligation of an isolated patent arterial duct.

**Setting**—Academic referral centre.

**Methods**—A preoperative transthoracic and transoesophageal echocardiogram was performed in theatre to look for aortic regurgitation. Thereafter, aortic flow was monitored throughout the operation by colour flow mapping with the transoesophageal probe in situ. Onset of aortic regurgitation was documented. An immediate postoperative transthoracic echocardiogram was performed on all patients and then daily until discharge on day 5. A follow up clinical and echocardiographic assessment was performed six weeks post-operatively.

**Results**—With ligation of the patent arterial duct, transoesophageal echocardiography showed immediate regurgitation in seven of the nine patients, seen as a small central jet on colour flow mapping. Six of the seven patients continued to have aortic regurgitation on transthoracic echocardiography before leaving theatre. In none was aortic regurgitation audible clinically. At discharge, five patients still had evidence of aortic regurgitation; of four seen at follow up six weeks later, only one had residual regurgitation.

**Conclusions**—Ligation of the patent arterial duct results in the acute termination of the "run off" in a volume overloaded situation. This, together with a rise in the peripheral vascular resistance and the persistence of increased proximal vascular capacitance, is considered to be the underlying aetiology of the acquired aortic regurgitation.

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**Keywords:** ligation of patent arterial duct; aortic regurgitation; echocardiography

Regurgitation of the tricuspid, mitral, and pulmonary valves in normal subjects has been documented previously by means of colour flow Doppler.<sup>1-4</sup> It has been noted, however, that normal children seldom if ever have aortic regurgitation on high resolution colour flow Doppler assessment.<sup>2,3</sup>

During the immediate follow up period after occlusion of a patent arterial duct, either surgically or by means of transcatheter umbrella closure, we noted that many of the patients had developed aortic regurgitation on colour flow mapping, which had not been present before ductal occlusion. The aortic regurgitant leak was seen as a narrow central jet extending a variable distance into the left ventricle. In none of the patients who underwent ductal occlusion by means of the transcatheter technique was the aortic valve crossed during the procedure. Similarly, the aortic valve was obviously not interfered with during surgical ligation of the duct. The aortic regurgitation was not clinically audible. It would not have been detected if routine colour flow Doppler had not been done after occlusion of the patent arterial duct. We now report the occurrence and duration of aortic regurgitation, noted at echocardiography, after ligation of a patent arterial duct in seven of nine children.

## Methods

The patient cohort consisted of nine children, each with an isolated patent arterial duct, who were referred for surgical ligation. Their ages ranged between 7 and 66 months (median 29 months), and their weights ranged from 3.7 to 17 kg (median 10 kg). All patients had an initial preligation transthoracic echocardiogram in theatre, using a 2.5 MHz or 5 MHz transducer and a Siemens 1200 echocardiography imaging system. They were already intubated and anaesthetised for surgery. A careful inspection for aortic regurgitation by means of colour flow mapping and Doppler interrogation was carried out. The size of the duct was also determined and ranged from 4 to 10 mm (median 5 mm). Thereafter, a Siemens paediatric single plane (11 mm) phased array transoesophageal transducer was introduced and positioned at the level of the aortic valve. Once again, a thorough check for aortic regurgitation was done. The aortic flow was then continuously monitored throughout the operation by means of transoesophageal echocardiography. The time taken from ductal ligation to the onset of aortic regurgitation was noted. Following ligation, the transoesophageal transducer was removed and the patients then underwent another transthoracic echocardiogram in theatre before transfer to the cardiothoracic intensive care unit. Thereafter, transthoracic echocardiograms were repeated postoperatively at 24 hours, 48 hours, 72 hours, and then again on day 5 before discharge, to document whether

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aortic regurgitation was still present. Patients were finally assessed at six weeks by clinical evaluation and transthoracic echocardiography.

### Results

The nine patients acted as their own control group in that preoperatively none had aortic regurgitation documented by echocardiography. Seven of the nine patients developed aortic regurgitation as soon as the duct was clamped. This was seen with transoesophageal echocardiography as a small central regurgitant jet on colour flow mapping. In all patients the systemic blood pressure rose immediately with ligation of the duct. However, the aortic regurgitation persisted despite manoeuvres by the anaesthetist to normalise the blood pressure.

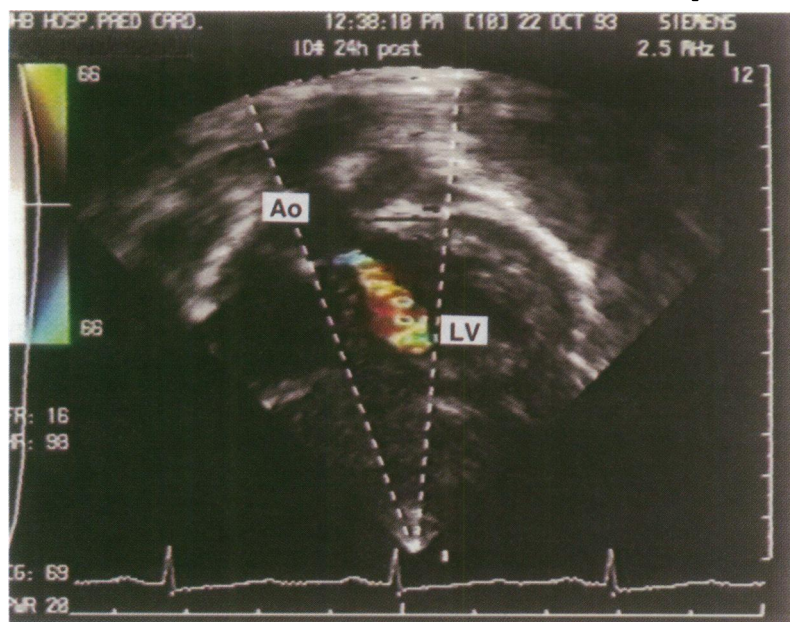


Figure 1 Aortic regurgitation demonstrated with transthoracic echocardiography 24 hours after ligation. Ao, aorta; LV, left ventricle.

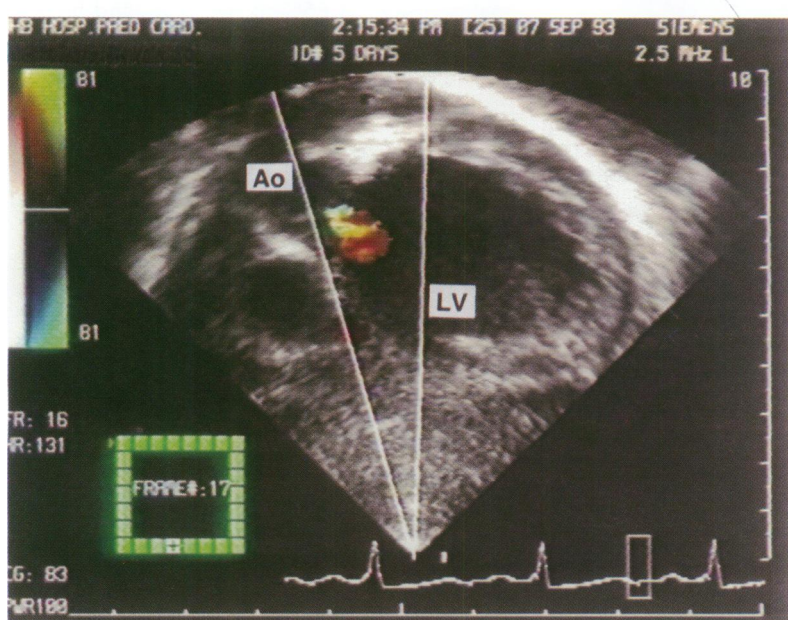


Figure 2 Aortic regurgitation seen with transthoracic echocardiogram five days after ligation in a second case. Ao, aorta; LV, left ventricle.

Of the seven patients with aortic regurgitation, six continued to show ongoing regurgitation with transthoracic echocardiography in theatre before transfer to the intensive care unit. Thereafter, daily transthoracic echocardiograms continued to show aortic regurgitation (fig 1) and by day 5, five of the seven patients still had evidence of residual incompetence on colour flow mapping (fig 2).

Of the five patients with documented aortic regurgitation at discharge, four were followed up six weeks later, and one patient continued to have an aortic regurgitant jet with colour flow mapping.

### Discussion

Recent studies have made use of colour flow mapping and Doppler echocardiography in the assessment of valvar flow in the normal population.<sup>1-3</sup> Yoshida and coworkers found that mitral, tricuspid, and pulmonary regurgitation are common in the normal population but aortic regurgitation did not occur in anyone.<sup>1</sup> Kostucki *et al* found aortic regurgitation in 33% of 25 healthy adults. The regurgitant jets did not exceed 50 cm/s.<sup>4</sup> However, this study was done solely with pulsed wave Doppler. Mattos *et al* found similar aortic regurgitant signals in two cases from a normal population of 25 children.<sup>2</sup> They felt that their findings did not represent true regurgitation because of the very low velocities. Brand and coworkers, on the other hand, were unable to detect aortic regurgitation in a cohort of 461 children with normal hearts.<sup>3</sup>

Doppler recordings in the descending aorta display only transient diastolic retrograde flow in normal children. Ward and Sholler showed that in conditions associated with "run off", such as a patent arterial duct, there is a pandiastolic retrograde flow in the descending aorta.<sup>5</sup> Of interest in their study was the finding that pandiastolic retrograde flow in the descending aorta persisted in a few cases following occlusion of the patent arterial duct. This may offer some explanation for our findings, although no mention was made of aortic valve flow patterns by the authors. They suggest that the systemic vasculature proximal to the duct increases in capacitance in response to chronic volume overload. With occlusion, the run off is suddenly removed and the increased regional capacitance permits transient continuation of the retrograde diastolic descending aortic flow. We feel that the run off in patients with a patent arterial duct lowers the systemic vascular resistance. With sudden occlusion, this run off is removed, causing the systemic vascular resistance to rise dramatically with resulting acute elevation of the systemic pressure (including the diastolic pressure), thus producing aortic regurgitation. The regurgitation persists for a number of days and, in one case, for up to six weeks, suggesting that readjustment of the systemic vascular resistance and vascular capacitance takes time to return to normal. The clinical significance of this transient aortic regurgitation is questionable. Nevertheless further studies looking at the mechanisms of aortic



incompetence following ductal occlusion are needed. The factors to be considered would include the size of the duct, continuous blood pressure monitoring, and direct measurements of systemic vascular resistance.

- 1 Yoshida K, Yoshikawa J, Shakudo M, Akasaka T, Jyo Y, Takao S, *et al.* Color Doppler evaluation of valvular regurgitation in normal subjects. *Circulation* 1988;78:840-8.
- 2 Mattos SdaS, Severi R, Cavalcanti CV, Freire M da F, Filho

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- 4 Kostucki W, Vandenbossche J, Friart A, Englert M. Pulsed Doppler regurgitant flow patterns of normal valves. *Am J Cardiol* 1986;58:309-13.
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## STAMPS IN CARDIOLOGY

### Christiaan Neethling Barnard (1922–)

South Africa issued a set of two stamps in 1969 to commemorate the first heart transplant and the 47th South African Medical Association Congress. The 2½ cent stamp depicts Professor Barnard and the Groote Schuur Hospital (A). A further stamp celebrating this event was issued in May 1991 as part of a four stamp series marking South African achievements between 1961 and 1991 (B). Christiaan Barnard also appears on the Paraguayan stamp from 1968 (C).

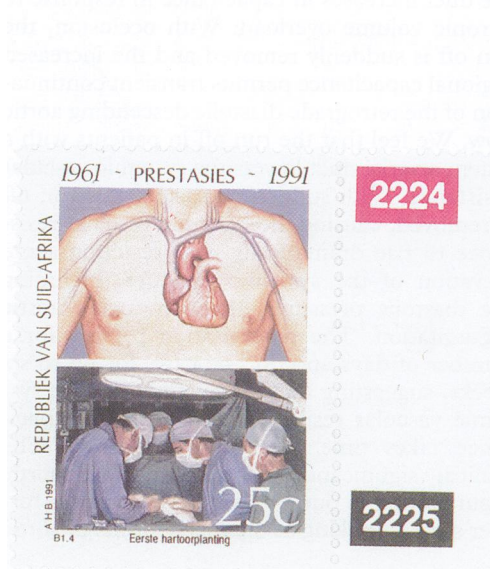
Christiaan Neethling Barnard performed the first human heart transplant at the Groote Schuur Hospital, Cape Town on 3 December 1967. Louis Washkansky, a 53 year old man, received the heart of a 25 year old woman and he lived for 18 days after the operation. His sec-

ond patient was a dentist, Dr Philip Blaiberg, who received a transplant on 2 January 1968. He lived for one year and seven months and was the first good medium term survivor. Although surgeons around the world followed Barnard's lead the results generally were dismal with 60% dead by the eighth postoperative day. This led to a moratorium on the procedure until cyclosporin transformed the scene when its value was established by R Y Calne in 1979 and was first used in heart transplantation in the early 1980s. This compound is a fungal metabolite which was discovered in a soil sample by Sandoz as a result of their search for new antibiotics. Although its antimicrobial activity was poor they had a far-sighted screening programme for other effects and this showed its value in immunosuppression. That important soil sample was not found by accident. Sandoz employees were asked to bring back soil samples from wherever they went on holiday or work and this one came from a desolate highland plateau in southern Norway called the Hardanger Vidda. Dr Norman Shumway of Stanford and Palo Alto is honoured as the surgeon who did the most, by means of long and careful research, to ensure that cardiac transplantation became the success that it now is. To date there have been about 25 000 heart transplants and the survival rate, usually with good quality of life, of 65% at five years and 50% at 10 years is encouraging.

M K DAVIES  
A HOLLMAN



(A)



(B)



(C)